

## BASL: Oral Presentations—Thursday 9 September 2010

### Clinical hepatology

#### OP01 ALCOHOL AND LIVER DISEASE DETECTION STUDY ALDDES: EARLY RESULTS AND IMPLICATIONS

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N Sheron, N Sheron, M Moore, P Roderick, W O'Brian, G Leydon, C Bowerman. *University of Southampton, UK*

**Introduction** Liver deaths have increased 5–8 fold in 30 years and the majority are alcohol related. Cirrhosis develops silently and presents late; around 25% of subjects die before they have a chance to stop drinking. Blood tests in routine use do not stage fibrosis and diagnosis often requires a liver biopsy—impractical for the 2 million UK residents drinking at levels that may result in cirrhosis. Newer blood tests detect cirrhosis and progressive fibrosis at an earlier stage and, combined with alcohol screening, could greatly improve detection and management of these patients, reducing morbidity and mortality.

**Aim** Assess feasibility of screening a primary care population for hazardous drinking using the AUDIT questionnaire, practicality of screening heavy alcohol users for liver disease using a non-invasive test and resource implications of assessment and follow-up.

**Method** We offered postal screening to 10 000 adults age 25–55 randomly selected from general practice lists using the AUDIT questionnaire. An offer of liver fibrosis tests was made to those screening positive. Those with marked elevation of fibrosis markers are referred on for liver health checks markers. Participants were followed up after 1 year.

**Results** 10 000 AUDITs were dispatched from 9 primary care sites. Response rate was 3677 (37%) Of these responders 907 were hazardous/harmful drinkers (18.5%), of whom 207 (4.2%) were dependent drinkers. Audit positive subjects were invited to attend a research clinic of whom 290 (32%) attended and were screened using our traffic light algorithm (HA, P3NP, PTS, albumin, INR) of whom 28 (9.6%) were red (cirrhosis/severe fibrosis) and 121 (41%) amber (50% likelihood of progressive fibrosis in a secondary care population).

**Conclusion** Postal screening for hazardous drinking followed by non-invasive liver assessment is feasible in the primary care setting. Ten percent of hazardous drinkers had good evidence of significant liver disease and a half had equivocal serum fibrosis markers, with these results fed back to subjects to encourage behaviour change. Further details and follow-up results will be available.

#### OP02 PREVALENCE AND SEVERITY OF NONALCOHOLIC FATTY LIVER DISEASE IN A LARGE PROSPECTIVE PRIMARY CARE COHORT WITH ABNORMAL LIVER FUNCTION TESTS

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M Armstrong, D Houlihan, L Bentham, J Shaw, S Olliff, J Neuberger, R Lilford, P Newsome. *Centre for Liver Research, University of Birmingham, UK*

**Introduction** An unexpected finding of abnormal liver function tests (ALFTs) is common in primary care. Currently there is a lack of data for determining the severity of liver disease, in particular nonalcoholic fatty liver disease (NAFLD), in the primary care setting.

**Aim** To determine the causes of unexpected ALFTs in a large primary care cohort. To determine the prevalence and severity of NAFLD in this cohort.

**Method** We analysed patients presenting with ALFTs, in the absence of known liver disease, to 8 primary care practices in Birmingham between 2006 and 2008. NAFLD was diagnosed in subjects with

fatty liver on ultrasound (USS), negative liver aetiology screen, and alcohol consumption of  $\geq 21$  &  $\geq 14$  units/week in males and females, respectively. As a sub-study we calculated the NAFLD Fibrosis Score (NFS) (Angulo, Hepatology 2007) to estimate the presence of advanced liver fibrosis (F3/F4 Kleiner classification) in subjects who met the diagnostic criteria for NAFLD.

**Results** Data from 1118 adult patients were analysed: 56% male; 83.9% white race; Age 60 years (48–70); 23.5% type 2 diabetes; 52.2% hypertension; body mass index 28.7 kg/m<sup>2</sup> (25.5–33.1) (values expressed as median (IQR) or %). Regarding alcohol consumption, 42.5% were non-drinkers, 32.2% drank within UK guidelines (male=21, female=14 units/week) and 26.3% drank in excess.

Liver aetiologies identified included alcohol-induced liver disease (25.7%), haemochromatosis/carrier (1.1%), viral hepatitis B/C (0.91%), primary biliary cirrhosis (0.8%), alpha-1-antitrypsin deficiency (0.18%) and primary sclerosing cholangitis (0.18%). Aetiology remained unexplained (normal USS, negative liver aetiology screen and alcohol consumption within UK guidelines) in 479 subjects, although at least 18% of this group had 2+ factors of the metabolic syndrome.

NAFLD was diagnosed in 26.4% (295/1118) of cases. A high NFS ( $>0.676$ ) was found in 7.6% of these cases indicating the presence of advanced liver fibrosis. The presence of advanced fibrosis could not be confidently excluded in 35.2% of NAFLD patients who had an “indeterminate” NFS ( $-1.455$  to  $0.676$ ), indicating the need for further investigation. Although NFS was validated in a secondary care cohort and may over-read severity in primary care, it is currently the best validated non-invasive method of assessing liver fibrosis in patients with NAFLD.

**Conclusion** NAFLD accounts for over a quarter of patients with ALFTs in primary care (26.9%). Of the patients with NAFLD a significant proportion either have advanced fibrosis (7.6%) or require further investigation (35.2%). This highlights the growing burden of NAFLD, and the need for validated methods of assessing NAFLD disease severity in primary care.

#### OP03 EFFECTS OF TWO YEARS OF LIRAGLUTIDE TREATMENT ON FATTY LIVER DISEASE IN PATIENTS WITH TYPE 2 DIABETES: ANALYSIS OF THE LIRAGLUTIDE EFFECT AND ACTION IN DIABETES-2 EXTENSION TRIAL

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<sup>1</sup>M Armstrong, <sup>1</sup>A Falahati, <sup>2</sup>D D Houlihan, <sup>1</sup>B Elbrand, <sup>1</sup>W E Schmidt, <sup>1</sup>S Gough, <sup>1</sup>P N Newsome. <sup>1</sup>Centre for Liver Research, University of Birmingham, UK; <sup>2</sup>Oxford Centre for Diabetes, Churchill Hospital, UK

**Introduction** Currently there are no established treatment options for fatty liver disease. Glucagon-like peptide 1 (GLP-1) analogues have been shown to reduce hepatic steatosis and markers of liver inflammation in rodents.

**Aim** To determine the efficacy of 2 years treatment with 1.8 mg liraglutide, a once-daily human GLP-1 analogue, on fatty liver disease and body composition in patients with poorly controlled type 2 diabetes (T2D).

**Method** Analysis was performed on the “Liraglutide Effect and Action in Diabetes-2” (LEAD-2) study cohort. LEAD-2 was a 26-week, double blind phase III trial with a 1.5-year open-label extension. Patients were randomised (2:2:2:2:1) to liraglutide 1.8, 1.2 or 0.6 mg/day, glimepiride 4 mg/day or placebo, all in combination with metformin 1.5–2 g/day. DEXA (n=160) and computerised tomography (CT) (n=154) sub-studies were performed to measure body fat composition and hepatic steatosis (defined by a liver-to-spleen attenuation ratio  $<1$ ), respectively.

Repeated measure and ANCOVA analysis was performed using last observation carried forward on the intention-to-treat population to estimate change from baseline. Values expressed as mean (SD).